A CORRELATIVE STUDY OF CLINICAL PRESENTATIONS AND ELECTRO ENCEPHALOGRAPHIC ABNORMALITIES IN CASES OF PARTIAL EPILEPSIES

THESIS FOR
DOCTOR OF MEDICINE
(MEDICINE)

BUNDELKHAND UNIVERSITY
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1992 Bharat Bhushan Kathuria

CERTIFICATE

This is to certify that the work entitled "A CORRELATIVE STUDY OF CLINICAL PRESENTATIONS AND ELECTROENCEPHALOGRAPHIC ABNORMALITIES IN CASES OF PARTIAL EPILEPSIES ", which is being submitted as a thesis for M.D. (Medicine) examination, 1992 of Bundel Khand University Jhansi has been carried out by Dr. Bharat bhushan Kathuria under my direct supervision and guidance. The techniques embodied in this work were under taken by the candidate himself. The results and observations recorded were checked and verified by me from time to time.

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This is to certify that the work entitled "A CORRELATIVE STUDY OF CLINICAL PRESENTATIONS AND ELECTROENCEPHALOGRAPHIC ABNORMALITIES IN CASES OF PARTIAL EPILEPSIES ", which is being submitted as a thesis for M.D. (Medicine) has been carried out by Dr. Bharat bhushan Kathuria in the Department of medicine, M.L.B. Medical College, Jhansi. He has put in the necessary stay in the Department of medicine as per University regulations.

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INTRODUCTION

INTRODUCTION

Epilepsies are one of the common neurological disorders which are described in ancient Ayurvedic literature as Apasmara. The word epilepsy is derived from greek words meaning to "seize upon" or " taking hold of".

Epilepsies are an important health problem and second common neurological disorders with a prevalence rate of around 9/1000 in India. So at a given a time, there are about 10 million epileptics in India.

The problem of epilepsy is not simple, because of usually long duration of treatment, attached social stigma and reduction in the working capacity of patient.

By and large the diagnosis of epilepsy is mainly clinical. Epileptology was only at preliminary stage before fifties of this century. But advent of EEG, CT scan and newer antiepileptic drugs has made it compulsory for a Physician to differentiate the idiopathic epilepsies from symptomatic epilepsies and generalised from localization related epilepsies,to ensure precise diagnosis,management and prognosis.

Various attempts have been made by experts to classify seizures and epileptic syndromes.Till date there is no Ideal classification.Most of these classifications are complex,time consuming and require ictal and inter-ictal EEG

recordings.Another difficulty is that the terminology of epilepsies has changed substantially but older terms like grand mal epilepsy and petil mal epilepsy are used by a large section of physicians today.

The various classification given by International league against epilepsy (ILAE) of 1969,1981.1985 and proposed revision of 1989 are being accepted all over the world.

Recently newer investigations like position emission tomography (PET), single photon emission computed tomography (SPECT), Magnetic resonance imaging (MRI) and sophisticated EEG related techniques have facilitated the diagnosis and aetiopathogenesis of some epileptic syndromes.

Facilities of newer investigations are not avaliable in most of the hospitals and EEG remains the mainstay for diagnosis of localisation related epilepsies.

In India higher incidence of partial epilepsies is noted as compared to western countries,probably because of greater frequency of CNS infections and birth injuries.

In partial eplepsies speecific type and pattern of seizure varies with locale of lesion Partial Seizures are classified as simple partial seizures,complex partial Seizures and Secondarily generalised partial seizures.Epileptiform EEG activity has been reported in 82% cases of partial epilepsies on routine EEG

(Hughes & Greener 1985).EEG May express a partial epileptic seizure by the appearance of repetitive activity from one region which is dissimilar to its background rhythm and which is not due to change in alertness. Attenuation of spontaneous activity has been described as representing seizure onset.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

1) Historical aspects

Epilepsy has the longest medical history (Hock & knight 1947) it was recognised in India before the 10th century b.c. (Joshi, 1971) Epilepsy is defined as "Apasmara" in ancient Indian medical texts. The prefix "Apa" meaning negation orloss & "Smara" meaning recollection of consious. charak described epilepsy as "paroxysmal loss of consiousness due to disturbance of memory and understanding of mind attended with convulsive seizures.

Epilepsy has been described in greek litrature as early as fifth century BC Famous persons in world the Alexander the crest, Julius caeser, st paul napolean, lard byron & Maupassant had epilepsy.

By careful pathological & clinical correlative studies hughlings jakson was first to interpret various manifestations of psycho motor seizures on a sound anatomical basis. He was one of the first to stress that psychic phenonmena are as much manifestations of epileptic discharge as the more spectacular tonic & clonic centractions.

Nomenclature

Various terms has been used, see has falling disease, fits, convulsions, seizure & epilepsy. The term 'Epilepsy' derived

from greek words meaning "to seize upon" or taking hold of. Asit is not a single disease, but a group of diseases manifesting in a common way, correct word nowadays in practice is epilepsies or epilepsies and epileptic syndromes'.

To define epilepsies precisely is not easy. Epilepsy are said to be a "tendency to recurring epileptic seizures" (Pefield & Fricksen 1941) it does not seems sufficient. To define epilepsies as paroxysmal cerebral dysarrhy thmia is also in sufficient. Hugling Jackson described epilepsy as recurrent seizures due to intermittent derangement of nervous system as a result of sudden excessive disorderly discharge of cerebral neurons. Best possible definition of epilepsies given by dichter (1987) is "Epilepsies are a group of disorders chara cterised by chronic recurrent paroxysmal alterations in neurological function caused by abnormalties in the electrical activity of brain".

CLASSIFICATION

As a basis to any discussion on classsification of epileptic phenonmena we need to be clear about distinctions between classification of epileptic seizures as opposed to those which apply to epilepsies themselves. With seizures we are primarily concerned with clinical manifestations and with their neurophysiological basis,whereas with epilepsies we are more concerned with grouping of seizures or syndromes,their natural histories and their underlying causes. Failure to observe these distinctions has often led to confusion in the past and still seems to be doing so, therefore at present time both of below

mentioned classifications (classification of epileptic seizures 1981 and classification of epilepsies 1989) are relevent.

CLASSIFICATION OF EPILEPTIC SEIZURES

(Modified from classification of I.L.A.E.1981)

PARTIAL SEIZURES (Focal,Local)

1. Simple partial seizures :

   - With motor signs
   - With somatosensory or special sensory symptoms
   - With autonomic symptoms or signs.
   - With psychic symptoms.

2. Complex partial seizures (with impairment of consciousness)

   - simple partial onset followed by impairment of consciousness
   - with impairment of consciousness at onset.

(a) - With impairment of consciousness only.

(b) - With automatism.

3. Partial seizures secondarily qeneralised.

GENERALISED SEIZURES :

(1) Non-convulsive seizures :

(a) Absence seizures.

(b) Atypical absence seizures.

(c) Myoclonic seizures.

(d) Atonic seizures.

(2) Convulsive seizures -

(a) Tonic -clonic seizures.

(b) Tonic seizures.

(c) Clonic seizures.

UNCLASSIFIED EPILEPTIC SEIZURES.

CLASSIFICATION OF EPILEPSIES AND EPILEPTIC SYNDROMES

(Modification of ILAE 1985 classification proposed in 1989 by commission on classification and terminilogy of ILAE.)

A. LOCALISATION RELATED (LOCAL,FOCAL,PARTIAL) EPILEPSIES AND SYNDROMES :

(1) Idiopathic (with age related onset)

- Benign childhood epilepsy with centrotemporal spikes
- Childhood epilepsy with occipital paroxysms
- primary reading epilepsy.

(2) Symptomatic :-

- Chronic progressive epilepsia partialis continua.
- syndromes characterized by specific modes of Preeipitation
- Temporal lobe epilepsies.
- Frontal lobe epilepsies.
- Partietal lobe epilepsies.
- Occipital lobe epilepsies.

(3) Cryptogenic (cause not known)

B. GENERALISED EPILEPSIES AND SYDROMES.

(1) Idiopathic :

- Benign neonatal familial convulsions.
- Benign neonatal convulsions.
- Benign epilepsy in infancy.
- Childhood absence epilepsy.
- Junvenile absence epilepsy.
- Junvenile myoclonic epilepsy.
- Epilepsy with generalised tonic clonic seizures (GTCS) on awakening.
- Other generalised idiopathic epilepsies.
- Epilepsies with seizures precipitated by specific modes of activation (Reflex eplepsies).

(2) Cryptogenic

- West syndrome.
- Lennox Gastaut syndrome.
- Epilepsy with myoclonic astatic seizures.

(3) Symptomatic

(i) Non-specific aetiology :

- Early myoclonic encephalopathy.
- Early infantile encephalopathy with suppression bursts.
- Other symptomatic generalised epilepsies.

(ii) Specific syndromes

- Epileptic seizures complicating other disease states.

(C) Epilepsies and syndromes undetermined whether focal or generalised.

(1) With both generalised and focal seizures.

- Neonatal seizures.
- Severe myoclonic epilepsy of infancy.
- Epilepsy with continuous spike and waves during slow wave sleep.
- Acquired epileptic aphasia.
- Other undetermined epilepsies.

(2) Without unequivocal qeneralised or focal features.

(D) Special syndromes

- Situation related seizures
- febrile convulsions.
- Isolated seizures or isolated status epilepticus.
- Seizures occurring only with acute metabolic or toxic events.

The important modifications in 1989 classification of epilepsies and epileptic synodromes proposed by ILAE are

1- Differentiation between the term "idiopathic" and "Cryptogenic" and inclusion of latter subtype. Idiopathic epilepsy has no underlying cause and is defined by age related onset,clinical and electroencephalographic characteristics and a presumed genetic etiology. The Term cryptogenic refers to a disorder whose cause is hidden or occult.

2- Inclusion of primary reading epilepsy as an additional entity in Localisation related Idiopathic epilepsies and syndromes.

3. inclusion of chronic progressive epilepsia partialis continua in the Localisation related symptomatic epilepsics and syndromes.

4. Specific mention of anatomical Cortical seizure Localisation in subgroup Localisation related symptomatic epilepsies and syndromes.

5. Another important inclusion is the category of epilepsies with seizures precipitated by specific mode of activation. The classic example of this group is reflex epilepsies for example-Hotwater epilepsy,eating epilepsy,Startle epilepsy.

EPIDEMIOLOGY

The over all prevalence rate of epilepsy in general population varies from as low 3-4 per 1000 to as high as 10.5 per thousand.Large variations in prevalence rates are attributed mainly to different criteria for selection of cases.A higher prevalence rate in developing countries is expected due to increase in birth trauma and infections.

In vast majority of studies males tend to predominate presumably due to more frequent head injuries.The commonest age of onset of epilepsies is 0-4 years.The incidence rates are higher within first decade.,somewhat lower in second decade and

then become low.The lowest prevalence rates which usually occur in first decade,increase in second decade and show a decline after 50 years.

AETIOPATHOGENESIS

Li and Jasper (1961) have clearly demonstrated that the epileptic process consists fundamentaly of hyperactive and hypersynchronous neuronal discharges.In chronic epilepsies the recurrent neuronal paroxysyms that underlie ictal events are transient expression of a more permanently physiologically disordered cortex.

The typical interictal EEG spike and wave complex reflects the summation of synchronised abnormal neuronal membrane potentials consisting of large paroxysmal depolarization shifts followed by prolonged after hyperpolarization.The depolarization shift results in enchanced neuronal excitation,while after hyperpolarization represents inhibition that may prevent ictal episode.Neurones in the cortical areas surrounding an epileptic focus may demonstrate paroxysmal hyperpolarization only forming an inhibitory zone,which appears to prevent epileptic spread during interictal period.

Despite knowledge of neuronal defects that can destabilize the membrane or interfere with balance of excitatory and inhibitory synaptic activity,fundamental mechanisms underlying spontaneous recurrent seizures in epilepsies remain to be unknown.The neuronal basis of seizure termination is mainly due

to self activating inhibitory machanisms rather than neuronal exhaution.

Site of origin of generalised tonic-clonic seizures is accepted as thalamic intralaminar system and mid brain spreading to cerebral cortex in bilaterally symmetrical and synchronous manner manifested in EEG as generalised spike and wave pattern. This condition has been termed " Corticoreticular epilepsy ".

In partial epilepsies an epileptic discharge can be assumed to be originating from a particular part of cerebral cortex.In some of the partial epilepsies,epileptic discharges though originating in one hemisphere is reflected as mirror image focus in the contralateral homologous area due to spread through commissural connections.The amplitude of the mirror image focus is usually smaller.So in cases of partial epilepsies,EEG findings are confirmatory in localisation of epileptic foci when findings are limited to specific lobe which is unusual.

Secondary generalization of partial epilepsies occurs through reticulothalamic corticalpathway of spread or through wide spread of discharges to same hemisphere from epileptic foci and spread via commisural connections to contralateral hemisphere.

In cases of complex partial seizures 90% of seizures originate from temporal lobe, usually medial aspect of temporal lobe.In remaining cases frontal lobe is commonly involved. Disturbance in consciousness favours involvement of

centrocephalic region.

Inhibitory neurotransmitter gamma amino butyric acid (GABA) may be a natural anticonvulsant found in the brain.GABA and acetyl choline have opposite effects upon neuronal excitability and an imbalance between these two substances could be predisposing factor for seizure activity (Jurgelsky and Thomas,1966).

AETIOLOGY

Commonly combination of cerebral insult and a genetic predisposition determines the appearance of epileptic seizures.

Genetic factors may contribute in three ways:

(i) An individual may inherit a low threshold for seizures as in reactive seizures i.e. benign febrile convulsions of infancy and childhood.

(ii) Specific primary epileptic conditions (Primary epilepsies) autosomal dominant genetic traits has been identified i.e. childhood and juvenile absence epilepsies ,partial sylvian epilepsy.

(iii) Secondary epilepsies i.e Myoclonic epilepsies,phenyl ketonuria.

ACQUIRED FACTORS

These include congenital lesion, head trauma, infection of brain and its coverings, brain tumours, systemic toxic and metabolic disorders, mesial temporal sclerosis, birth trauma.

The diagnostic role of inter-ictal EEG in epilepsies has been questioned by many authors (Goodin, 1984). Diagnosis of epilepsy is mainly clinical and role of EEG is restricted to confirmation of provisional diagnosis, to classify the seizure disorder, localisation of epileptic foci and guiding prognosis (insome cases) (Githhchley, 1978). In a study by Goodin (1964) 60% of epileptic patients in general population had positive epileptiform activity on EEG. While only 4% of non epileptic has positive EEG.

LOCALISATION RELATED EPILEPSIES AND SYNDROMES

1. Benign childhood epilepsy with centro-temporal spikes.

This type occures in 15-20% of epileptics in the age group of 3-13 year, more commonly in males (60%). Seizure pattern consists of brief spells of simple partial hemifacial motor seizures, frequently during sleep (70%), with somatosensory onset and tendency to tonic, tonic-clonic seizures of facial and neck muscles, manifesting as speech arrest and drooling of saliva, seizure, occures usully once in 2-12 months. EEG changes consists of blunt high voltage centrotemporal spikes often followed by slow waves usually precipitated by sleep.

It is an autosomal dominant gene with age dependent penetrance . Monotherapy with phenytoin or carbamazepine is successful. Usually good recovery occures before 15-16 years of age.

2. Childhood epilepsy with occpital paroxysms.

Onset is usually between $1_{1/2}$ years to 8 years, usual symptoms (amaurosis in 65%, Scotoma in 55% , hallucinations in 25%, illusions in 10% cases) predominate, nocturnal partial seizures (usually hemiclonic) and post ictal headache (30%) are fairly common.

Paroxysmal high voltage spike wave and/or sharp waves localised in the occipital or posterior temporal region over one or both hemispheres occur rhythmically on EEG.

These finding tend to disappear after 9 years of age. Prognosis is favourable regardless of age, seizures are well controlled in 60% cases. Drugs useful are phenytoin, carbamazepine and primidone.

3.Benign partial epilepsy with frontal foci:

Partial seizures usually begin between 4-8 years of age. The seizure pattern is of adversive type with head deviation, simple absence seizures may precede or coexist. EEG findings consists of unilateral or bilateral frontal foci. In about 50% cases 3 Hz spike and wave complexes are observed, EEG abnormalities normalize before the age of 13 years.

4. Primary Reading Epilepsy

Symptomatic:

Chronic progressive epilepsia partialis continua :

It is seen in young children with unilateral chronic cerebral inflammatory disorderes or in adults after severe anoxia and major cerebrovascular accident The continous partial motor seizures usually involving face reflect multiple rather than single lesion . This form is often unresponsive to medications. Seizures may disappear after about 1-2 years in about one third of cases.

Temporal lobe epilepsies :

This term is not synonymous with complex partial seizures, psychomotor and limbic seizure. The term temporal lobe epilepsies means that a temporal lobe focus in EEG is demonstrable. Symptomatology usually consists of an aura, psychic symptoms, alteration of consciousness automatisms and commonly secondary spread to manifest as tonic-clonig seizures.

Auras consists of experiential phenomena, which patient perceives as warning of impending seizure episode. Psychic symptoms consist of :

a. dysmnesic symptoms i.e. feeling of familiarity (de ja vu) and unfamiliarity (Jamais vu)

b. cognitive disturbances such as dreamy state,depersonalization and time distortion.

c. Affective symptoms,fear and rage,depression and elation and sometimes sexual excitation (orgasmic epilepsy).

d. Delusions and hallucinations.

Alteration of consciousness unsually reflects involvement of limbic structures and their connections. Autonomic auras such as epigastric distress or vomiting most commonly precede temporal lobe epilepsies which are associated with altered consciouness. Those seizures which are preceded by olfactory aura's are called uncinate seizures.EEG findings consist of unilateral or bilateral spikes.

Frontal lobe epilepsies.

Seizures pattern may consist of :

a. Tonic,clonic movements starting from discrete parts such as feet,hands and facial muscles and progressing leading to unilateral or bilateral involvement. When spread occurs in an orderly fashion along the precentral gyrus jacksonian march occurs.These seizures may be followed by post ictal weakness (Todd's paresis).

b. Complex versive movements in form of, turning of head,eyes and body towards one side and posturing of one or more extremities.

c. Adversive seizures : Turning of head and eyes away from epileptic focus and elevation of contralateral arm.It occurs with involment of supplementary motor cortex.

d. Inappropriate laughter associated with humour (Gelastic epilepsy).

e. Some times they may manifest as psychomtor seizures and atypical absence seizures.

EEG usually shows unilateral or bilateral spike wave complexes and sharp waves.

Parietal lobe epilepsies :

They usually manifest as localised paresthesia or numbness from onset of a seizure.Spread occurs readily to frontal cortex leading to sensory motor seizures.Post ictal phenonmena such as anaesthesia may follow after simple partial seizures involving parietal lobe.

Occipital lobe epilepsies :

The seizures pattern usually manifest as unformed luminous vision and amaurosis.

GENERALISED EPILEPSIES AND SYNDROMES

1. Benign Neonatal familial convulsions :

They usually occur on 2nd or 3rd day.Seizures pattern consists of clonic or apneic seizures.No specific EEG changes are observed.About 14% of these develop some form of epilepsy.

2. Neonatal convulsions.

They occur on 5th day of birth.Seizure pattern is of clonic or apneic type.Alternate sharp theta waves are observed on EEG.

3. Benign myoclonic epilepsy in infancy :

Age group involved is 1-2 years.Seizures pattern consists of breif spells of generalised myoclonus and brief bursts of spike wave complexes are observed on EEG.

4. Childhood absence epilepsy (Pyknolepsy)

It affects children 6-7 years of age,manifests as many absence seizures during a day.EEG findings consist of bilateral synchronous spikes and wave.Some of these children may develop Generalised tonic clonic seizures (GTCS) later on.

5. Juvenile Absence :(Petit mal).

It manifests as absence with retropulsive movements,seizure frequency is less than childhood absence.EEG findings show 3 Hz spike-wave bursts.This type of epilepsy has excellent response to therapy.

6. Juvenile myoclonic epilepsy :(Impulsive petit mal)

It manifests as bilateral single or repetitive arrhythmic irreggular myoclonic jerks in arms and shoulders.It may progess to generalised Tonic clonic Seizures associated with infrequent absences.EEG findings consist of precipitation of episode by photic stimulation,rapid generalised 4 to 6 Hz spike waves/ polyspike -wave. It affects about 7% of adolescent epileptic patients. Sodium valproate is the drug of choice.

7. Epilepsy with Generalised Tonic-Clonic Seizures on awakening :

It usually affects young adults. Familial occurence is

frequent.Seizure type consist of Generalised Tonic Clonic Seizures shortly after awakening (90%) or in the evening (10%) EEG findings consist generalised high voltage sharp waves following by slow waves.

8. Epilepsies with seizures precipitated by specific modes of activation :

Classic example of this group are reflex epilepsies which are generalised i.e. Hotwater epilepsy, eating epilepsy,tactile epilepsy,paroxysmal dystonic seizures.

Cryptogenic

1. West syndrome :

It affects infants usually in 4-7 months age group.Seizure type consist of salam attacks.EEG shows burst suppression with distorted background activity.Prognosis is poor, even after treatment with steroids.

2. Lennox Gastaut Syndrome :

Childern of 1-8 year of age group show this picture consisting of more than one variety of generalised seizures,presence of mental retardation and generalised sharp and slow wave complexes of less than 3 Hz and some times sleep bursts of fast rhythm.

Refractoriness to common anti-epileptic drugs is the rule.

3. Epilepsy with myoclonic astatic seizures :

Age of onset is usually 7-12 years.Frequently male Children are affected.Seizure type consist of myoclonic akinetic absence with clonic and atonic components.Frequently these patients may land into status epilepticus of generalised tonic -clonic type.Regular fast spike wave and polyspike wave abnormalities are noticed on EEG.

Epilepsies and Syndromes undetermined whether focal or generalised

1. Epilepsy with continuous spike wave activity during sleep.

Seizure type consist of Generalised tonic clonic seizures during sleep and atypical seizures. Observed EEG findings consist of continuous diffuse spike-wave during sleep.

2. Acquired epileptic child hood aphasia (Landau Kleffner syndrome) Seizure type consist of verbal auditory agnosia with secondarily generalised partial motor seizures. EEG findings consist of multi focal spikes and spike wave complexes. Course is benign usually with complete remission before the age of 15 years.

EEG in the Diagnosis of epilepsy

Definitive diagnosis of epilepsy requires demonstration of ictal EEG changes simultaneously with occurrence of seizure,which is rarely possible except in patients with absence seizures,juvenile myoclonic seizures and partial seizures with minor motor seizure.

Due to paroxysmal nature of the disease a normal EEG does not exclude epilepsy.The probability of positive EEG findings is related to several factors.

a. Duration of recording.

b. Wake-sleep study.

c. Activation procedures used.

d. Times of EEG recording.

In a single routine EEG,positive findings are usually observed in around 60% cases with sleep deprivation, but if 3 EEG recordings have been taken epileptiform changes may be observed in 90% cases.

e. Use of additional electrodes.

f. Anticonvulsant therapy.

EEG abnormlities in epilepsies :

They can be grouped in three catagories-

1. Non-specific abnormalities

2. Specific abnormalities

3. Background abnormalities

1. Non-specific abnormalities

a) 3 Hz Delta paroxysms

i. Frontal intermittent rhythmic Delta activity (FIRDA).

ii. Occipital intermittent rhythmic Delta activity (OIRDA)

b) Runs of bilaterally synchronous delta.

c) Bursts of bilaterally synchronous theta.

d) 8-12 Hz activity of higher amplitude than the background alpha activity.

2. Specific epileptiform abnormalities.

a. Spikes
b. polyspikes
c. High voltage sharp waves
d. spike-wave complexes.
e. polyspike-wave complexes
f. sharp wave discharges.

3. Background abnormalities.

a. mild degree of diffuse slowing while on anticonvulsant therapy.

b. Diffuse marked slowing-may occur during post ictal period for few hours to 1-2 days.

c. background slowing other than above usually correlates with mental subnormalities.

Following newer investigation have facilatated the diagnosis of epilepsies.

A) NEUROPHYSIOLOGICAL INVESTIGATIONS :

1. Routine EEG
2. Ambulatory cassette recording
3. EEG telemetry
    a. Cable telemetry.
    b. Radio telemetry.
4. EEG recording with closed circuit television by spilt screen technique.
5. Computrised EEG neuromapping.
6. Magnetic encephalography.

B) NEURORADIOLOGICAL PROCEDURES :

1. Plain radiography.
2. CT scanning.
3. MRI imaging.
4. Isotope encephalography
5. PET scanning.
6. SPECT scanning.

EEG abnormalities reported in partial epilepsies are sequential spikes and sharp waves,Sinusoidal waves Rhythmic waves and attenuation of spontaneous activity.In a study designed to asses EEG morphology of partial epileptic seizures. Warren T Blume et al (1984) studied 66 consecutive patients with elecrographic partial seizures.Different EEG phenomenon noted were

1. Attenuation,sinusoidal waves.
2. Spikes,spike-waves,polyspike-waves,sharp waves,saw toothed waves.

The second group was reffered as repetitive epileptiform potentials (REPs).

They noted : Total partients = 66

Sinusoidal waves in 20 patients (30%)

REPs in 17 patients (25%)

Both in 29 patients (45%)

ONSET AND MORPHOLOGY OF EEG CHANGES

Onset	Evolution
Sinusoidal (41)- Sinusoidal only 31	Sinusoidal only 20
	REPs appear (11)
REPs - REPs only -25	REPs only 17
	Sinusoidal appear-8
Both (sinusoidal & REPs) -10	

Spikes and sharp waves were the most common repetitive epilepti form potentials enountered.

Attenuation of spontaneous activity with onset of seizure was noted in 11% of cases. Gibbs (1964) reported 80% incidence of temporal foci inclinical psychomotor epilepsy.

Many workers have tried to develop methods of deriving components of local activity in certain area. Bo Hjorth (1976) made an important contribution by formulating source sensing derivations connected between EEG amplifiers and recording channels,derivations of this type constitute better description of local activity than do the conventional,common reference and bipolar derivations.

The model implied that the observed surface pattern is a secondary effect of primary field components perpendicular to surface.

Smith et al(1985) have demonstrated the reliability of a non invasive method for successfully localising the local components of scalp EEG.Dipole localisation method (DLM) is a computer assisted,mathematical method based on electrical field theory.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

Clinicial manifestations of epilepsies are varied and diagnosis of specific type of epileptic state or syndrome is based mainly on history,clinical features, and EEG.

Role of surface EEG is thus restricted to differentiate epilepsy from other causes of seizures,to substantiate the clinical diagnosis of epilepsy,to classify seizure disoreders,localising the epileptogenic focus and guiding the prognosis.

In a developing country like India,sophisticated facilities helpful in localisation of epileptogenic source (CT Scan, MRI, PET, SPECT) are available in few centres only.Further a large section of population of developing country like India can not bear the expenses for these investigations.In this context EEG is very cheap and available in most of medical college hospitals,Considering this fact present study was conducted with following aims and objectives :

1. To assess the prevalence of partial (localisation related) epilepsies amongst all epilepsies.

2. To Assess the frequency of different types of localisation related epilepsies.

3. Evaluation of EEG abnormalities in localisation related epilepsies.

4. Study of clinical manifestations of different types of localisation related epilepsies.

5. correlation of specific EEG abnormalities with different types of localisation related epilepsies.

6. To assess the role of scalp EEG in localising the site of epileptogenic focus.

MATERIALS AND METHODS

MATERIAL AND METHODS

The patients suffering from seizure disorders who attended the out patient departments of Medicine,pediatries,Neurology and Psychiatry.of Maharani Laxmi Bai Medical College,Hospital Jhansi from Jan.1990 to Jan.,1991 formed the sample of the study.Patients who required investisation or having uncontrolled seizures were admitted.Patients of both sexes and all the age groups were included in the study.

METHODS :

All the patients were evaluated on following lines.

Clinical History

1. General History with special emphasis on detail of seizure disorder was taken.

2. Clinical examination,specially of nervous system was performed and a record of clinical symptomatoloty with clinical examination was maintained.

Investigations

1. Routine EEG,

EEG, utilizing 10-20 system of electrode placement with both referential and bipolar recordings,after overnight sleep deprivation with at least 3 minutes of hyperventilation and photic stimulation was recorded for at least 30 minutes on 8 channel EEG machine.

2. Radiological :

I. X-ray skull A.P.skull A.P.and lateral view.

II. X-ray chest PA view(whenever relevent)

III. Computerised axial tomography of head if possible.

3. Fundus examination.

4. Cerebrospinal fluid examination

5. Biochemical tests

a. Serum calcium.

b. Blood urea,blood sugar.

6. V.D.R.L. test.

History of seizure disorder was taken from patient and relatives of partient.Special emphasis was given to :

i. Age of onset of seizures.

ii. Frequency of seizures.

iii. Time of last seizure episode.

iv. Predisposing and aetiological factors.

v. Family history of seizures.

vi. Precipitating factors.

vii. Generalised or partial seizures.

viii. Premonitory symptoms.

ix. Pre-ictal, ictal and post ictal events.

x. Specific abnormalities.

All the patients were specifically evaluated for the above mentioned data.

General examination and examination of nervous system was interpreted for associated disease with seizure disorder,associated neurological deficit and evidence of localising neurological deficit and evidence of localising value of seizure.

EEG was interpreted using EEG diagnostic criteria given by kiloh et al (1982).regarding presence of

1. Background rhythm
2. Clear paroxysmal (epileptiform events on EEG)
3. Slow wave abnormalities.
4. Sequential spikes,spikes and waves originating from a focus.
5. Symmetry.
6. Presence of rhythmic waves.
7. Attenuation of spontaneous activity at onset of seizure.
8. Sinusoidal waves.
9. Specific abnormalities.

Other investigations,which were required were done in each case. CT scan was done whenever possible.

EEG data obtained from above observations was correlated with different types of localisation related (partial)epilepsies and comparison with similar observations were made.

In the end,findings were tabulated and analysed.

OBSERVATIONS

OBSERVATIONS

Present study was carried out on 196 patients presenting with complaint of seizure/seizures who attended the out patient department of medicine, Paediatrics, Neurology and Psychiatry of Maharani Laxmi Bai Medical college Hospital Jhansi from Jan 1990 to Jan 1991.

Amongst 196 epileptics, 9(4.6%) cases were of unclassified epileptic seizures :-

Distribution of classifiable cases :-

(a) Seizure type :- Primarily generalised seizures were present in 108 cases (55.10%).79 cases were of parcial seizures. Secondarily generalised partial seizures were present in 36 cases followed by simple partial seizures (14 cases)(table I)

Table I:Showing Clinical types of Seizures.

Types of Seizures	No. of cases	Percentage
(A) Generalised Seizures.	108	55.10%
Tonic-clonic	95(87.96%)	
Tonic	2(1.85%)	
Atonic	3(2.77%)	
Myoclonic	2(1.85%)	
Absenses	4(3.70%)	
Atypical Absenses	1(0.92%)	

(B) Partial Seizures	79	**40.40%**
(i) Secondarily Generalised	36	18.36
(ii) Simple Partial	29	14.79
(iii) Complex Partial	14	7.14
Unclassified epileptic Seizures	9	**4.59**
Total	196	100.00%

(b) Age of onset :-

In this study age of onset of seizures was highest (48%) in the first decade of life. In 92% cases seizures started below the age at 20 years (Table II)

Table No.II:Showing Age of onset of Seizures

Age Group	No. of cases	Percentage
0 - 10	94	47.95%
10 - 20	86	43.87%
20 - 30	8	4.08%
30 - 40	3	1.53%
40 - 50	3	1.53%
50 - 60	2	1.02%
Total	196	100.00%

Out of 196 cases 138 were male and 58 female.Thus Male to Female ratio was 2.38/1. Male predominence has been noted in earlier studies also.

Features of Partial epilepsies

Nearly 40% of patients in this study presented as Partial Seizures.Age of these patients ranged from 6 months to 52 years Male to Female Ratio in these cases was 2.56/1. Out of these cases,45.5%(36cases) presented as Secondarily generalised Partial Seizures,17.72%(14 cases) presented as Complex partial Seizures and 36.71(29 cases) presented as Simple Partial seizures. (Table No. III)

Table III :- Various types of partial Seizures

Type of Partial Seizures	No.of cases	Perecntage
(A) Simple Partial Seizures	-29	36.7%
(B) Complex Partial Seizures	-14	17.7%
(C) Secondarily generalised Partial Seizures	-36	45.6%
Total No. of cases	79	100%

Frequency of Seizure episodes varied from single episode in a year to several episodes per day.13 patients had only one seizure episode at the time of EEG recording.7 Patients were admitted in status epilepticus.One patient presented as chronic epilepsia partialis continua.

Predisposing factors could be worked out in 19 cases. Head injury was the commonest cause (9 cases),followed by Birth anoxia - 5 cases and intracranial space occupying lesion (3 cases). Inflammatory brain disease was present in 2 cases.

Precipitating factors were present in 12 cases in form of sleep - 5 cases, sleep deprivation 3 cases, fatigue 2 cases and fever in 2 cases.

Out of 79 cases of partial seizures abnormal EEG findings were found in 58 cases. In 4 cases EEG findings were generalised without any focal changes. In the rest 54 cases hemispherical assymetry on EEG was noticed. Abnoraml EEG findings were confined to one hemisphere in 16 cases. Commonest EEG findings in Partial Seizures were sharp waves.

Features of Simple Partial Seizures

Amongst total 196 cases,29 cases(14.8%)had simple partial Seizures.Age of patients ranged from. 10 Months to 52 years.Male to Female Ratio was 2.2 :1.In more than half of cases seizures started below the age of 10 years.Predisposing factors could be detected in 4 cases two patients had head injuries,one patient had intracranial space occupying lesion and one case had history of prolonged labour with forceps applications & birth anoxia.

One patient had facial palsy. One patient had right sided hemiparesis with difficulty in speech.

Seizures started on right half of body in 12 cases and left half of body in 16 cases. Postictal features were present in only 20 cases.Clinical features are sum marised in Table IV.

Table No.IV:

Clinical features of simple Partial Seizures.

Total No. of cases -29

Clinical features.	
(A) ONSET	
Right half of body.	
-- Motor	---- 7
-- Sensori Motor	---- 2
-- Sensory	---- 3
Left side of body.	
-- Motor	---- 9
-- Sensory	---- 5
-- Sensory Motor	---- 2
(B) ICTAL features	
Tonic	---- 13
Clonic	---- 9
Tonic-Clonic	---- 5
Autonomic	---- 2
(C) Postical features (20 cases)	
(1).Headache	---- 10
(2).Postictal Paresis	--- 4
(3).Drowsiness	---- 7
(4).Sleep	---- 3
(5) Vomiting	---- 2
(6).Generalised weakness	---- 3

Abnormal EEG findings were observed in 16 cases out of 29 cases.

Localisation of EEG abnormalties to a single cerebral hemisphere was possible only in 7 cases. Localisation wasnot possible in 2 cases. (Table V).

Parietal Lobe was the commonest site of epileptic foci (8 cases) (Table no. VII). EEG abnormalities consisted of sharp waves, spike with sharp waves, and high voltage sharp waves.

Commenest EEG abnormalities observed were repetitive epeleptiform potientials (REP's) observed in 11 cases out of total 16 cases in which EEG was abnormal (Table no. VI). Sharp waves were most common. High voltage sinusoidal waves were observed in 5 cases. These waves were generalised with high voltage in specific region in 3 cases and localised in 1 case. Focal slowing (in parietal region) was observed in one case. This patient had a seizure episode 12 hours back. Frontal intermittent rhythmic delta activity was observed in one patient aged 1 1/2 years with history of simple partial seizures after Head injury in frontal region one month back. EEG morphological characterstics had little correlation with different types of simple partial seizures but topographical distribution of EEG abnormalities correlated well with site & type of seizure onset.

CT scan was performed in one patient with history of simple partial seizures (motor) 10 months after head injury. CT scan was normal in this case.

Table No. V :
Showing EEG features of Simple Partial Seizures

EEG Findings	No. of cases	Percentage
(A) Normal	13	44.83%
(B) Abnormal	16	55.17%
1.Localisation possible	7	
Right Hemisphere	3	
Left Hemisphere	4	
2.Localisation with secondary generalization.	7	
Right Hemisphere	2	
Left Hemisphere	3	
Central Hemisphere	1	
Centroparietal	1	
3.Localisation not possible (Generalized)	2	

Table No. VI

Various EEG abnormalities in simple partial seizures

EEG findings	No. of cases
A) Specific epileptiform Abnormalties	14 cases
i) High voltage sinusoidal waves	5 "
ii) Sharp waves	6 "
iii) Localised spikes	1 "
iv) Spike wave	2 "
v) Sharp waves discharges	2 "
B) Nonspecific abnormalities Frontal intermittent Rhythmic Delta activity (FIRDA)	1 "
C) Background abmormalities Focal slowing	1 "

Table No.VII :

Epileptic foci in case of simple partial seizures

EEG Focus	No. of cases
1).Frontal	1
2).Parietal	3
3).Central	1
4).Centroparietal	1
5).Parieto temporal	2
6).Fronto temporal	2
7).Occipito parieto temporal	2
8).One Cerebral Hemisphere	2

Features of complex Partial Seizures

Out of 196 cases, there were only 14 cases of complex Partial Seizures. Age of Patients ranged from 7 years to 52 years. In 6 cases seizures started below the age of 10 years in the rest 8 cases seizures started after the age of 10 years. Family history was positive in one case. Predisposing factors could be detected in 3 cases only, two with history of birth anoxia and one with history of head injury. One patient was not performing well in his studies after seizures had started.

Out of 14 cases history of aura was present in 9 cases. Automatisms and motor phenenmenon were present in 8 cases. In 3 patients ictal episode progressed to generalised tonic-clonic

seizures while in two patients ictal episode progressed to generalised tonic seizures.

Post convulsive features were present in 10 cases in form of amnesia (6 cases) Headache 3 cases and vomiting in one. Table VIII summarises the clinical features in cases of complex partial seizures.

Table VIII :Showing Clinical features of Complex Partial seizures (Total no of cases -14)

Clinical Features	No.of cases	Percentage
AURA.	9	64.29%
Visual Hallucinations	2	
Semato sensory Hallucination	3	
Auditory Hallucination	2	
Vertigo	1	
De Javu	1	
Automatisms/Motor Phenomena	8	57.15%
Restlessness	3	
Lateral deviation of Head and eyes.	2	
Upward deviation of eyeballs	1	
Smacking of lips	1	
Tearing of clothes	1	
Amnesia	6	42.85%
Other associated features	5	35.71%
Generalised Tonic-Clonic Seizures	3	
Tonic spasms	2	

EEG Findings.

EEG abnormalities were observed in 8 cases out of total 14 cases of complex partial seizures.Rest 6 cases had Normal EEG.They consisted of temporal spikes,phase resersal and burst of sharp waves.Bitemporal spikes were present in 2 cases.

In three cases epileptic foci was unilateral without any generalized findings.In 3 cases findings were generalised with some focal change in one side EEG findings are summarised in table No. IX & X.

Table IX:EEG findings in complex Partial Seizures.

Normal	---	6	42.85%
Abnormal	---	8	57.15%
Unilateral temporal Spikes	-	3	
Bitemporal Spikes	---	2	
Temporoparietal	---	1	
Frontotemporal	---	1	
Centrotemporal	---	1	

Table X :- EEG findings in complex Partial Seizures.

EGG findings.		No. of Cases.	Percentage.
Normal		6	-42.85%
Abnormal		8	-57.15%
Focal	(1) Right -------	2	
	(2) Left -------	1	

(3) Bilateral ------	2	
Focal with secondarily genaralised		
(1) Right -------	1	
(2) Left -------	2	
Total No. of Cases	14	100.00

C T Scan could be performed in 2 cases only. In one case Bilateral temporal lobe atropy was present. Another C T Scan was normal .Other investigations were normal.

Features of secondarily Generalised Partial Seizures.

Out of total 196 cases studied 36(18%) cases were of secondarily generalised Partial seizures.Age of patients ranged from 1 year to 52 years .Male to female ratio was approximalely 2:1. Frequency of seizure episodes varied from one episode in a year to several episodes in a day.Seven patients had only single seizure episode at the time of recording.Three patients were admitted in status epillepticus.

Predisposing factors could be worked out in 9 cases.Head injury was commonest in 4 cases,followed by history of birth anoxia in 3 cases and intracranical space occupying lesion in 2 cases.Precipitating factors were present in 4 cases in form of sleep(2 case),fatigue and fever (1 case).

Premonitory symptoms were present in 16 cases.They include abnormal sensations,ghabrahat,feeling of muscle contractions in parts where seizures started and headache onset of seizures was from right half of body in 13 cases and from left half of body in 16 cases.In 7 cases patient or patient's attendants were unable to tell exactly the side involved first either due to single episode or due to lack of any person watching the patient at the time of seizures.Common parts of body from where seizure started were,great toes, hands,lips and eyelids.In nearly half of cases patient experienced some sort of abnormal feeling in the part of body from where seizures started well before the onset of seizure episode.Nature of seizure episode at onset was motor,sensory motor,sensory or autonomic.

Motor symptoms were in form of twiching,tonic spasms or clonic movements.Sensory symptoms were in the form of numbness and tingling sensations in the extremities.One patient had voiding of micturition at the onset of seizure and one had vomiting with abnormal sensation in epigastric regions.28 patients had unconsiousness,while 8 remained consious. Tonic - clonic movements were present in 30 cases while only tonic spasms in 6 cases.

Post convulsive symptoms were present in form of Headache,drowsiness,post ictal paresis,weakness and confusional states.

Clinical features of secondarily generalised partial seizures are summarised in Table No.(XI).

Table No.XI :

Clinical features of secondary generalised epilepsies (Total No. of cases 36)

Clinical features		No.of cases	Percentage
ONSET			
Side	Right	13	36.11%
	Left	16	44.44%
	Could not be confirmed	7	19.44%
Nature	Motor	16	44.44%
	Sensori motor	10	27.77%
	Sensory	8	22.22%
	Autonomic	2	5.55%
CONVULSIVE PHASE			
	Unconsiousness	28	77.78%
	No alteration of consiousness	8	22.22%
	Tonic-clonic movements	30	83.33%
	Tonic spasms	6	-16.67%
POSTCONVULSIVE PHASE.			
	Headache	10	
	Headache & Drowsiness	22	
	Postical Paresis	8	
	Generalised weakness	2	
	Confusional state	2	

Associated Neurological features were present in 10 cases which are summarised below in table XII.

Table No.XII :

Neurological features of Secondarily Generalised epilepsis.

Clinical	No. of cases
Mental Retardation	3
Hyperactivity	1
Optic Atrophy	1
Hemiparesies	3
Transient Aphasia	2
TOtal	10

EEG findings

Out of 36 cases of secondarily generalised partial seizures abnormal EEG was found in 29 cases (80.5%).In 4 cases EEG findings were generalised without any focal changes.In rest 7 cases EEG findings were bilaterally assymmetrical on two sides.In 18 cases findings were generalised with some focal changes on one side.

EEG findings were in form of high voltage sinusiodal waves, Sharp waves,Burst of sharp waves,phase reversal,spikes,spike and waves.

Table No.XIII :

EEG findings of secondarily Generalised Partial Seizures

(A) Normal	7		19.44%
(B) Abnormal	29		80.56%
(i) Generalised	4		
		R-3	
(ii) Focal	7		
		L-4	
(iii) Secondarily Generalised with focal	18		
-- Right Hemisphere	6		
-- Left Hemisphere	8		
-- Central	3		
-- Bilateral	1		

As is shown in Table XIII,29 cases showed abnormal findings.In 4 cases EEG findings were generalised and no focal changes were present.In 7 cases findings were confined to one or another hemisphere.

In 18 cases an epileptic focus was present with generalised epileptiform abnormalities on EEG. Among these findings were confined to right hemisphere in 6 cases,to left hemisphere in 8 cases,to central region in 3 cases and bilateral in one case.

In 25 cases an epilepitc focus was present.In nine cases epileptic focus involved one area of scalp.In 16 cases epileptic

focus was not exactly Localised and involved two areas of scalp. In four cases localisation of epileptic foci was not possible because changes were generalised. (Table XIV)

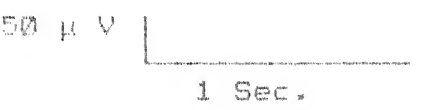
Table No.XIV :

Showing Focus in cases of secondarily generalised seizures

EEG Focus		
(1) Frontal	2	
(2) Parietal	4	
(3) Temporal	2	
(4) Occipital	1	
(5) Fronto parietal	2	
(6) Centro parietal	6	
(7) Occipito parietal	1	
(8) Parieto temporal	2	
(9) Occipito temporal	4	
(10)Bitemporal	1	
(A) Localisation possible (In above cases)	25	(69.45%)
(B) Localisation not possible	4 cases	
(C) Normal EEG	7 cases	
Total No. of cases	36	100.00%

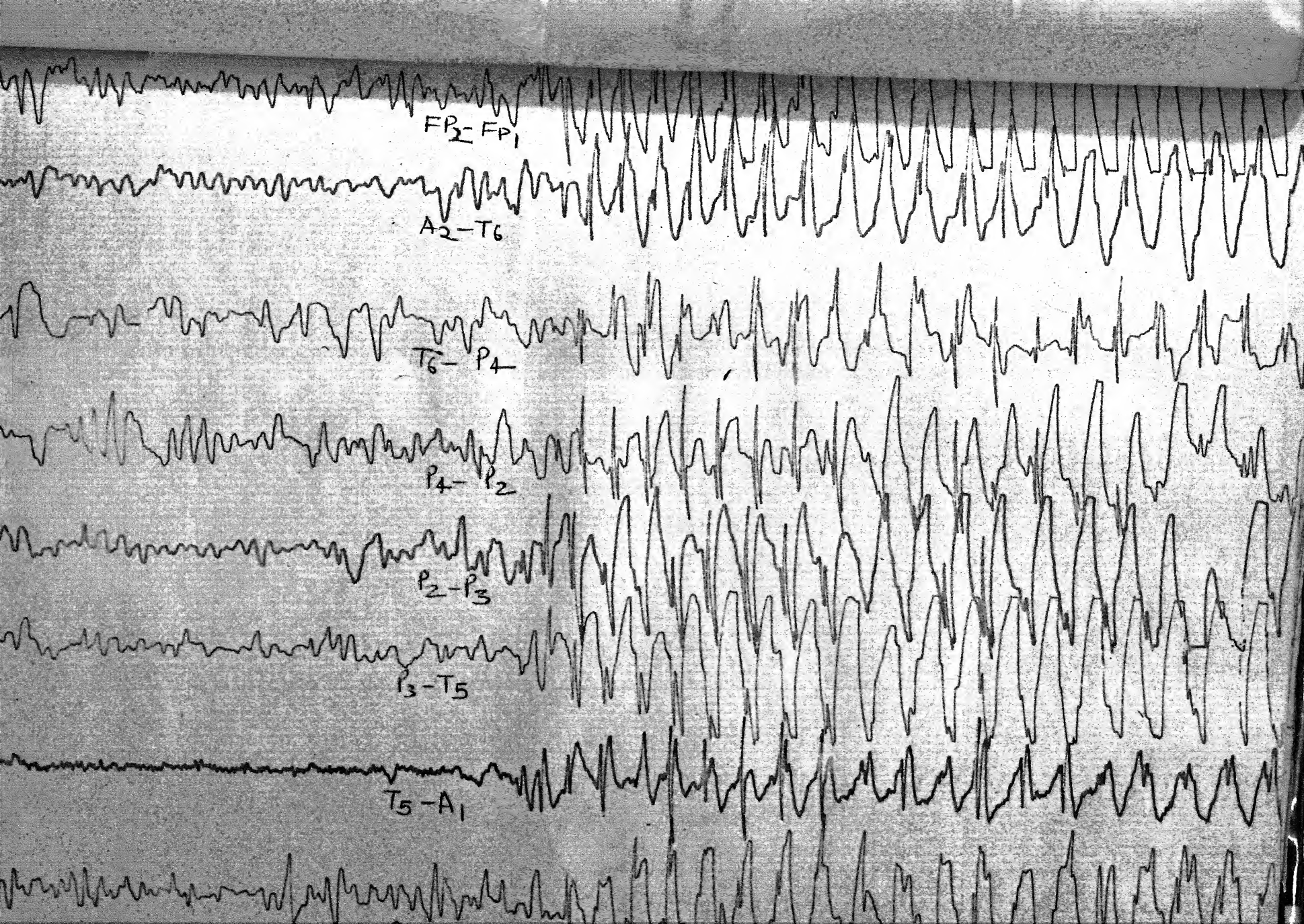
CT Scan was performed in 3 cases one patient showed a granuloma in left parietal region.In one case it showed a tuberculoma in central region. In rest of the cases CT Scans were normal. On examination of fundus of one patient optic atrophy was detected. X-Ray chest of one patient showed bilateral tubercular infiltration .X-Ray skull were normal. Other investigations were normal.

EEG No. 532 : Showing phase reversal, in the posterior temporal region




Fθ - T4
T4 - T6
T6 - O2
Fp1 - F7
F7 - T3
T3 - T5
T5 - O1

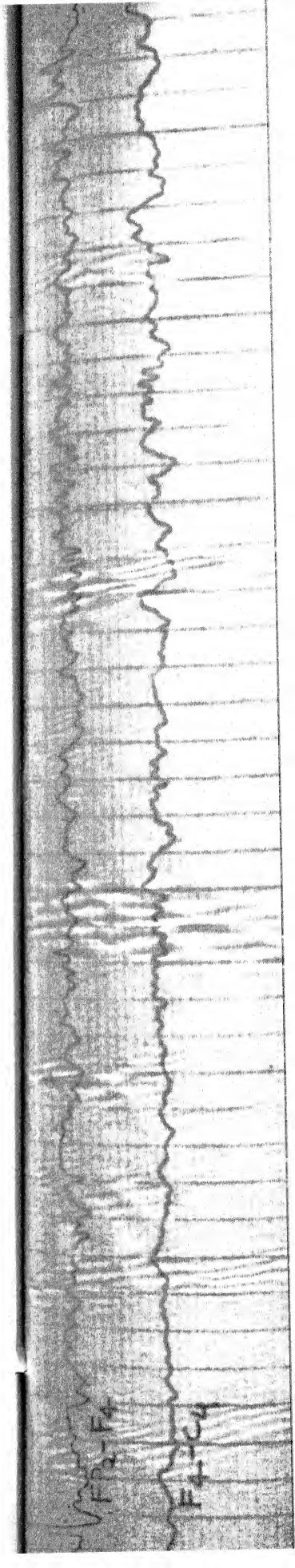
EEG No. 681 : showing generalised polyspikes and waves in a case of complex partial seizures

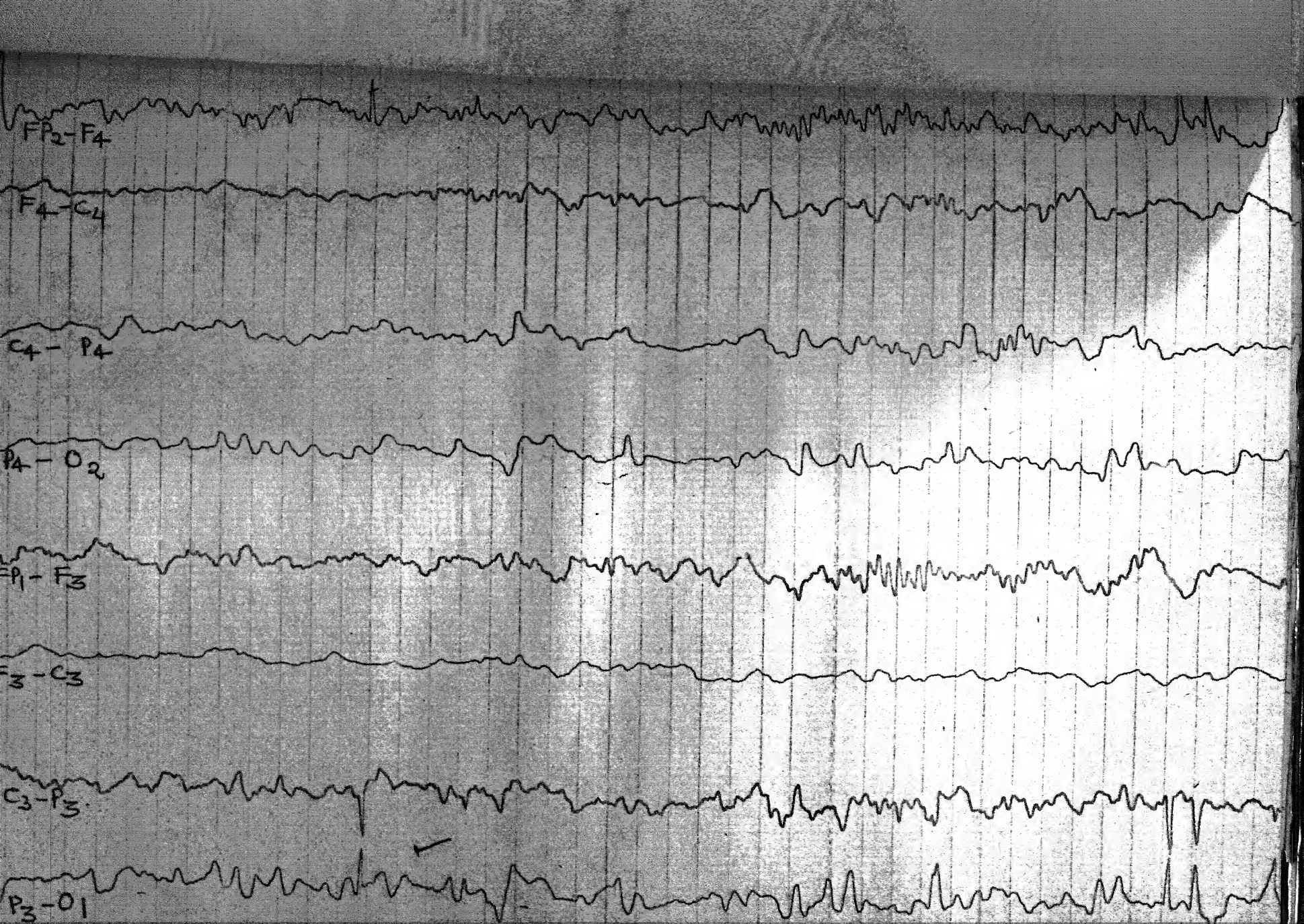

FP2-FP1
A2-T6
T6-P4
P4-P2
P2-P3
P3-T5
T5-A1

EEG No. 667 : Showing phase reversal over left parietal region.

50 μV

1 Sec.

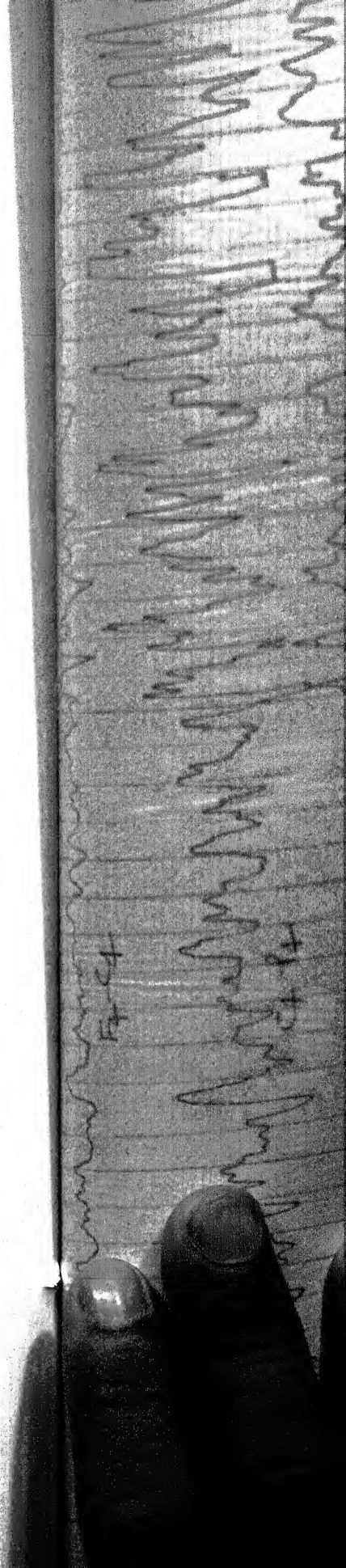


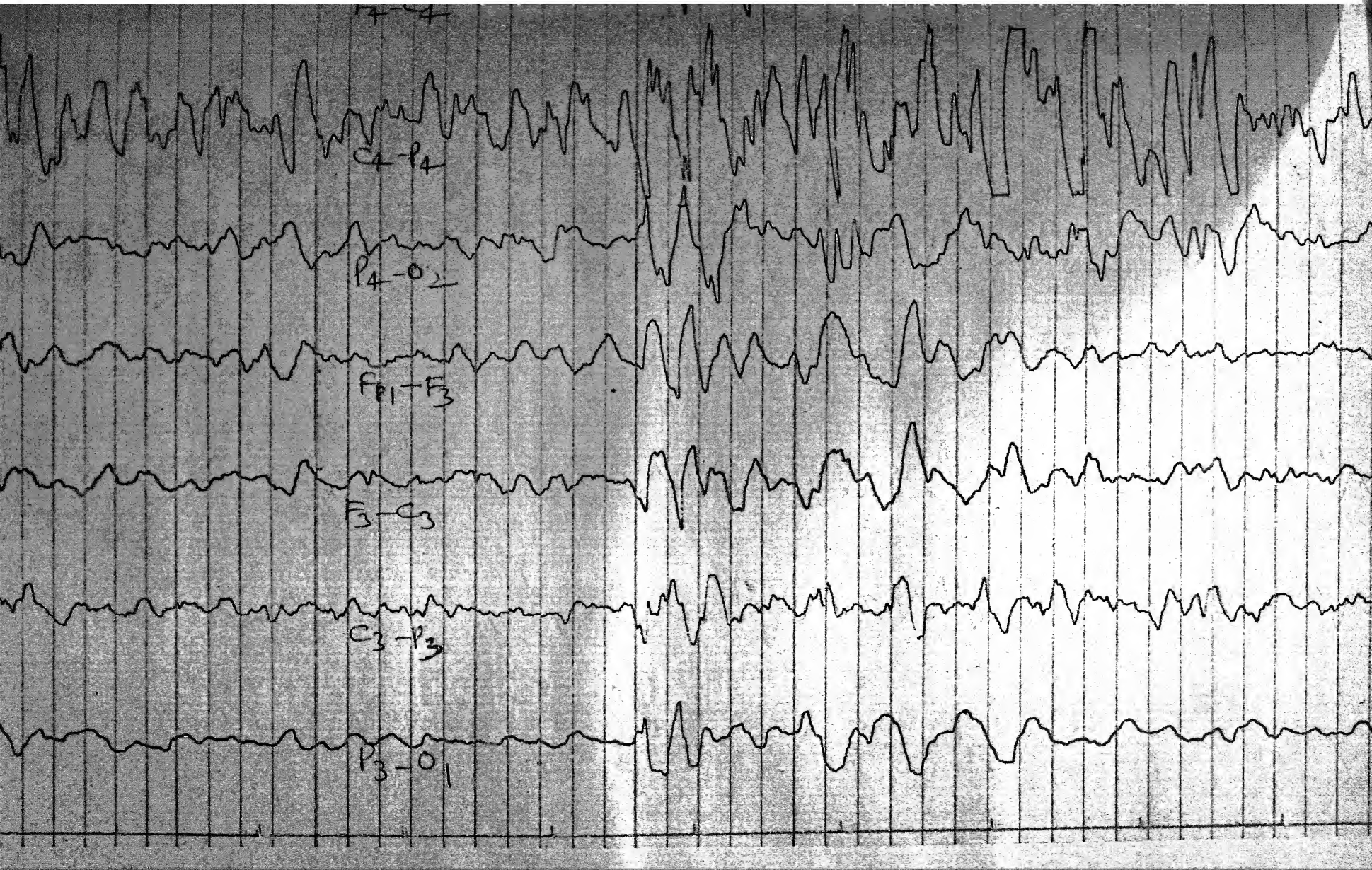

FP2-F4
F4-C4
C4-P4
P4-O2
P1-F3
F3-C3
C3-P3
P3-O1

G No. 612 : Showing secondary generalisation of epileptiform changes starting from right side.

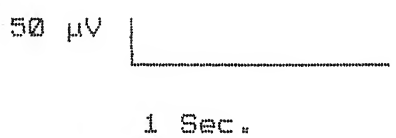
50 µV

1 Sec.



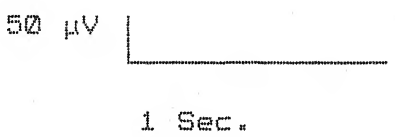

C4-P4
P4-O2
Fp1-F3
F3-C3
C3-P3
P3-O1

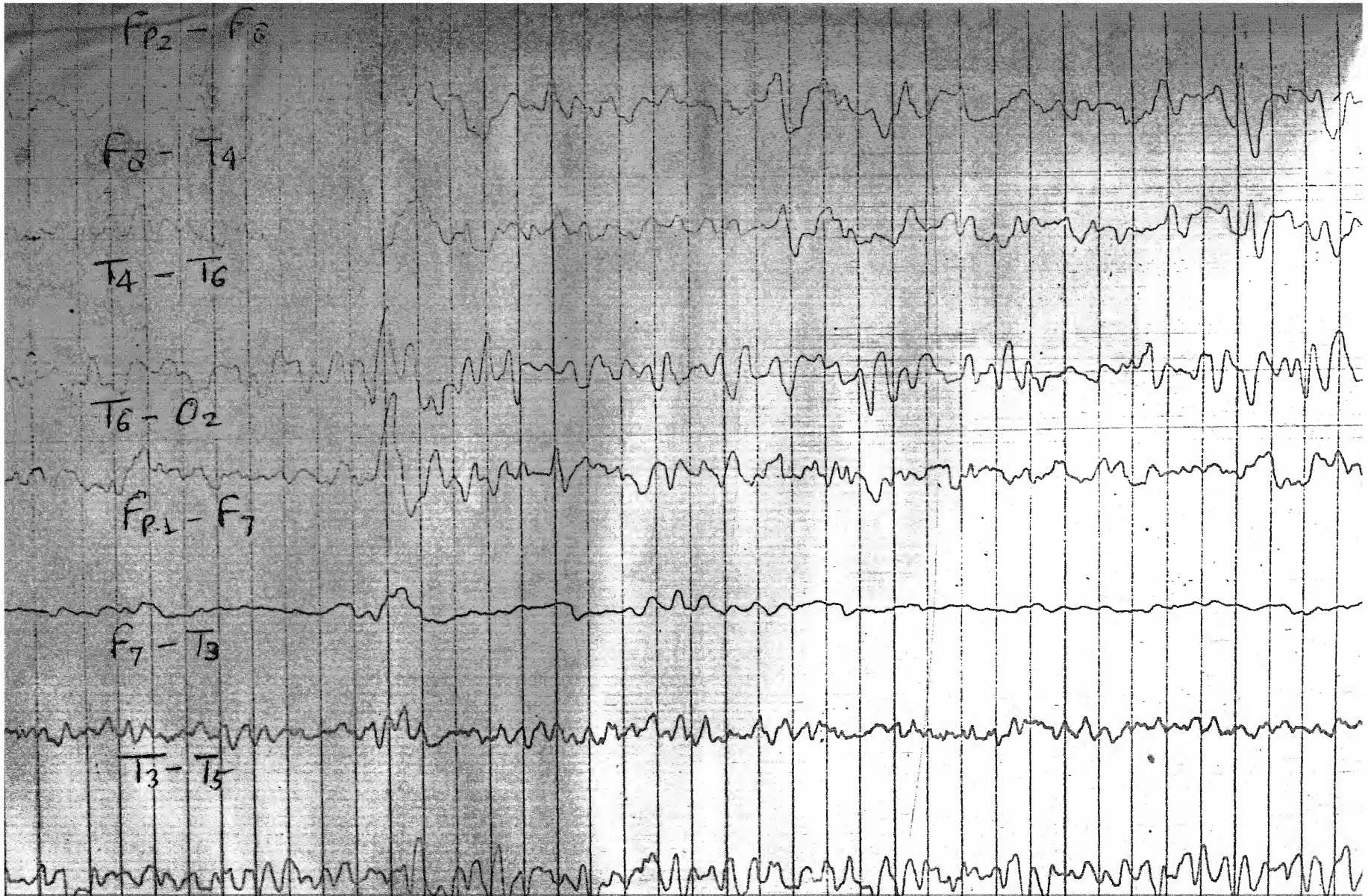
545 : Showing generalised epileptiform discharges with maximum amplitude over right parietal region in a case of secondarily generalised seizures.




A2 – T6
T6 – P4
P4 – P2
P2 – P3
P3 – T5
T5 – A1
O2 – O1

) No. 617 : Showing asymmetry of waves with phase reversal in right frontal region in a case of complex partial seizures.




Fp2 - F8
F8 - T4
T4 - T6
T6 - O2
Fp1 - F7
F7 - T3
T3 - T5

DISCUSSION

DISCUSSION

Epilepsy is a common illness. Partial epilepsies are frequent and EEG techniques are very much helpful for diagnosis of partial epilepsies. Considering this we have decided to conduct a correlative study of clinical presentations and EEG abnormalities in cases partial epilepsies.

Age : Age specific prevalence ratios for partial epilepsies have been found to be lowest in first decade (Brewis et al - 1966, Juul Jensen 1976 and Heirer et al - 1986). However in this study maximum number of cases belonged to third decade.

Age of onset of seizures was highest in second decade and slightly lower in first decade. Then it decreased substantially in fourth, fifth and sixth decade.

Sex : Approximately (70%) of patients were male. Male to female ratio was 2.4:1. Findings were almost similar to Dixit and Mishra (1987-88) and Sood and Mishra (1988-89). Male dominance in this study might be because of more attention given to male member of family in rural population of India.

In many studies males were predominant, male to female ratio being 1.4:1.

Clinical Seizure types :

In the present study seizures have been classified on the

basis of classification of epileptic seizures given by ILAE in 1981 and modified in 1982 by Marsden and Reynolds.

Gastaut et al (1975) studied 6000 epileptics and worked out their different classifiable groups and their relative frequency in children and adults. A similar study by Joshi et al (1977) had been carried out in India. Both these studies were based on classification almost similar to that used in the present study. Similar studies were done in this institution by Dixit and Mishra in (1987-88) and Sood and Mishra in (1988-89). Primary generalised seizures dominated our series (55%), in comparison to 30% in the series of Gastaut et al (1975), 20% in the series of Joshi et al (1977), 51.7% by Dixit and Mishra (1987-88) and 62% by Sood and Mishra (1988-89). Next common type was secondarily generalised partial seizures (18%). Figure was higher in comparison to 12% and 15% in Gastaut et al (1975) and Joshi et al (1977) respectively. Cases of simple partial seizures in the present study were (15%) as compared to 50% (Joshi et al, 1977) Smallest number of cases were of complex partial seizures (7.1) which was almost similar i.e. 7% in the study of Joshi et al (1977).

FEATURES OF SECONDARILY GENERALISED PARTIAL SEIZURES

In the present study, out of 196 cases, 36(18.36%) cases were of this type of seizures premonitory symptoms were present clinically, in 16 cases.

Six cases presented clinically as tonic spasms, while in 30 cases, clinical presentation was of tonic clonic types. In a study done by Dixit et al (1987-88) these cases were included in primary generalised epilepsy. In these particular cases, it is thought that the spread of focal discharges was so rapid, that focal changes were not present clinically but can be recorded in EEG.

Age of patients ranged from 1 month to 52 years. Marethan One third of patients were below the age of 10 years. Male to female ratio was 2.8:1. Frequency of seizures varied from several attacks in a day to single attack in a year.

Aetiological factor was present in 9(25%) cases, slightly more in comparision to primary generalised seizures. Head injury was commonest in 4 cases followed by history of birth anoxia in 3 cases and by intracranial space occupying lesion in 3 cases, inflammatory brain disease in 2 cases. In a series by Joshi et al (1977) aetiological factors were present in 60% of cases. The lesser percentage of etiological factors in the present study might be because CT scan could be performed in fewer number of cases, particularily in the suspected cases of interacranial space occupying lesions. According to Gastaut and Gastaut (1976), CT scans detect 20% more cerebral lesions than the combination of long established techniques like, x-ray skull, EEG and cerebral angiography.

Precipitating factors were present in 4 cases (11%) in the form of sleep-4 cases, fatigue - 1 cases, fever - 1 case.

Out of 48 cases, motor symptoms were present in 16 cases (44%). They were present in the form of twitching of angle of mouth, eye movement, clonic movements of limb and tonic spasm. Sensory symptoms were in the form of tingling and numbness sensation 8 (22%) cases. Sensory motor symptoms were present in 10(28%) cases. Autonomic symptoms i.e. epigastric pain were present in 5% cases.

EEG FINDINGS AND CLINICAL CORRELATION

EEG abnormalities were detected in about (80%) cases, percentage being significantly higher than in cases with primary generalised seizures. Findings were focal in 7(19.4%) without any secondary generalisation while in 18(50%) cases, secondary generalisation was present. In 4(11.11%) cases no epileptic foci could be detected and findings were generalised and bilaterally symmetrical and synchronous. In 12(33%) cases epileptic focus were on left side and right sided epileptic focus was present in 9(25%) cases. In three cases focus was central. In one another case it was bilateral in both temporal region. This patient was presented clinically with aura of epigastric pain and tonic-clonic convulsions.

Focus was clearly demonsrated in a single lobe area of scalp in 9(25%) cases. In all the cases focus determination was based

In 16(44%) cases, EEG focus involved two areas of scalp i.e. fronto-perietal-2 cases, parieto-occipital 1 case, centroparietal- 6 cases, and occipitotemporal 4 cases, Parietotemporal. EEG changes were spreaded in such a manner that exact localisation was not possible. This might be, because epileptic focus was deep and EEG changes involved two areas of scalp. One patient of head injury showed haematoma in frontal region and EEG showed fronto- parietal focus. Patient had memory impairment and some behavioural abnormality after head injury. One other interesting case, showed central EEG focus, as phase reversal with generalised epileptic discharge bilaterally symmetrical and syncronous. CT scan showed small granuloma in left mid central region. Patient presented clinically with right sided sensory motor, focal onset followed by generalised tonic-clonic convulsions and unconsciousness.

In one case foci were bilateral in both the temporal region. Patient presented clinically with autonomic symptoms as epigastric pain before tonic clonic convulsion and unconsciousness. This might be because of mirror foci in two lobes (Kiloh et al, 1982).

There were two patients of tonic spasm, one showed phase reversal in fronto-parietal region with generalised epilepic discharges. Other showed slowing in the fronto-parietal region, with bilateral generalised discharges.

In 4 (11%) cases, EEG showed epileptic discharges, bilaterally symmetrical and almost synchronous without any focal changes. The work of Tukel and Jasper (1952) and penfield and Jasper (1954) showed it was possible for bilaterally synchroncous abnormalities to appear in the EEGs of patients with epilepsy apparently caused by unilateral parasaggittal orbitofrontal, or anterior temporal lesions. They called this pattern of belaterally symmetrical and synchronous discharges due to some focal lesion as secondary bilateral synchrony.

FEATURES OF SIMPLE PARTIAL SEIZURES :

In our study, 29(15%) cases were of simple partial seizures. Other studies reported 20% simple partial seizures (Sharan, 1987), whereas 50% and 62% reported by Gastaut et al (1975) and Joshi et al (1977), respectively. The low percentage of simple partial seizures in the present study might be because in Bundelkhand region most of the persons are of rural society and uneducated, did not bother about simple twiching and minor movements in hand or feet without loss of consciousness. Age of the patients ranged from 10 months to 52 years. In approximately half of the cases, seizures started below the age of 10 years. Male to female ratio was 2.2:1.

Predisposing factor were present in (11%) cases. However, Joshi et al (1972) found predisposing factor in 30% of cases, whereas Dixit and Mishra (1987-88) could found predisposing

factor in 15.4% of cases. History of head injury was present in 2(8.69%) cases.

Out of 20 cases, seizures started from left side in 16 cases and from right side in 12 cases. Left sided dominance of symptoms could not be explained. Start of seizure from thumb or fingures could possibly be because representation of hand in brain involves much area and is more prone to injuries.

EEG FINDINGS AND CLINICAL CORRELATION

EEG abnormalities could be detected in 16(55.17%) cases, EEG focus involving the single area of scalp was found in 5 cases. One case showed central focus. In 5 cases, EEG focus involved two areas of scalp. In two cases multiple foci was present on one side and exact localisation of focus could not be determined. In two cases EEG focus involved occipito parieto temporal region.

In 7 cases, findings were strictly focal without any generalisation to other side. Whereas in other cases, there is generalisation of epileptic discharges to other side. This might be because epileptic discharges in EEG were spreaded bilaterally but clinically patient did not exhibit generalised features.

FEATURES OF COMPLEX PARTIAL SEIZURES

There were 14 cases (7.1%) cases of complex partial

seizures. male to female ratio was 2.6:1. In 7(53.85%) cases seizures startes below the age of 10 years. In 16 cases (76.9%) seizures started below the age of 20 years. Findings are almost similar to Aird et al (1967) who reported that 42% cases had their first attack in the first decade and over 75% in the first two decade of life. Visamani and Sawhney (1966) and Reddy (1971) drawn the similar conclusion.

Out of 14 cases, predisposing factors could be worked out only in 3(19.37%) cases in the form of head injury - 1 case and birth anoxia - two cases. This is almost similar with earlier studies done in this institution by Dixit and Mishra (1987-88) and Sood and Mishra (1988-89). Lesser percentage of aetiological factor was probably because of the smaller sample and in expected cases CT scans could not be performed.

Aura was presented in 9(64%) of cases. It is almost similar findings as compared to 60% of cases of Shukla et al, 1979.

Out of 14 cases, 3(21%) case also had tonic clonic convulsions. Several authors included these somatomotor manifestations as a clinical features of psychomotor seizures (Bossi et al, 1984).

EEG FINDINGS AND CLINICAL CORRELATION

57.11% cases showed EEG abnormality. Epileptic focus was

present in 8 cases viz. temporal - 3 cases, centrotemporal 1 case, temporo- parietal - 1 case, fronto-temporal - 1 case. In one case foci were bilateral in both the temporal regions and one might be mirror image of the other. Patient presented with sometosensory hallucination, smacking of lips and generalised tonic movements and unconsciousness.

SUMMARY AND CONCLUSIONS

SUMMARY AND CONCLUSIONS

The Present study entitled "A correlative study of clinical presentations annd electroencephalographic abnormaleties in cases of partial epilepsies" was carried out on 196 patients of various types of epilepsies A detailed history was taken from patients and relatives., Detailed physical examination with more stress on examination of nervous system was done in every case. Various laboratory investigations including routine EEG was done in every case. CT scans were done whenever possible. Observations were tabutated and analysed.

5% of cases were of unclassified epileptic seizures .In nearly half of cases seizures started in the first decade. Overall male to female ratio was 2.4:1 .Approximately 55% of cases belonged to the group of primarily generalised seizures, and 40% to the group of partial seizures. Amongst partial seizures, secondarily generalised partial seizures were most common 36 (45%) cases, followed by simple partial seizures 29 (36.7%) cases ,and complex partial seizures 14 (17.7) cases.

58 (74.3%) patients presented with history of recurrent seizures 13(16%) cases presented with history of only single seizre episode. 7(9%) cases presented as status epilepticus. One patient (1.26%) presented as chronic epilepsia partialis continua.

Age of patients ranged from 6 months 6 to 52 years. Seizure frequency was variable. In nearly half of cases seizures started below the age of 10 years. Predisposing factors were present in about 12(15%) cases they consisted of sleep, sleep deprivation fotigue and fever mainly. Premonitory symptoms were present in 31% of cases, mainly in cases belonging to the group of secondarily generalised partial seizures and complex partial seizures ictal events consisted of unconsiousness 31(39.2%) cases, motor phenonmen 55(69.6%) cases, Sensory involvement 20(25.3%)cases & autonomic features in 4(5%) cases.

Post ictal features were present in 38(48%)cases.They mainly consisted of Headache,drowsiness,postictal paresis,generalised weakness,confusional states and combination of these.

Associated neurological problems were present in 12(15%) cases.They consisted of mental retardation in 3 cases,hemiparesis in 3 cases,transient inability to speak in 2 cases,Hyperactivity in one case ,optic atrophy in one case,confusional state in one case and post ictal paresis in one case each.Respiratory system involvement was present in 2 cases in form of pulmonary tubercular infiltration(1 case) and primary complex in 1 case. EEG abnormalities were present in 53 cases of partial Seizures out of total 79 cases of partial Seizures,and in 56 cases of primarily generalised Seizures out of 108 cases of generalised Seizures.

Thus EEG abnormalities were observed in 67% cases of partial Seizures and in 52% cases of primarily generalised seizures.

CT scan could be performed in a limited number of cases(10 cases).It was abnormal in 3 cases with partial Seizures.In one case it should a parietal lobe granuloma on left side.In a single case a tuberculoma in central region was present.In Rest of cases CT scans were normal.

Following conclusions could be drawn from the present study.

1. Patients were of younger age group and seizures started below the age of 20 years in most of the cases.

2.Primarily generalised Seizures were commonest type of seizures (55%).Partial Seizures comprised nearly 40% of cases amongst all epilepsies.

3.Males predominated the sample as male to female ratio was 2.4 :1.

4. The comonest type of partial seizures were secondarily generalised partial seizures(45.6%) followed by simple partial seizures(36.7%)and complex partial seizures(17.7%).

5. Motor phenonmena were the commonest (69.6%)components of ictal events followed by sensory involvement (25.3%) and autonomic features(5%).

6. EEG abnormalities were present more commonly in partial seizures (67%) as compared to that in primarily generalised seizures (52%).

7. The common EEG abnormalities in partial seizures were,sharp waves,burst of sharp waves and high voltage sinusoidal waves.

8. EEG morphological characteristics had little correlation with different types of partial seizures(except complex partial seizures) but topographieal distribution of EEG abnormalities correlated well with site of seizure onset.

9. Localisation of epilepic foci to a specific region was possible in 17 cases out 79 cases(21.5%) on routine EEG.

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